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THE MECHANISM OF THE PRODUCTION OF HEART MURMURS

PART I

The mechanism of the production of both heart sounds and heart murmurs has been an object of speculation since the days of Laennec. Many problems remain still unsolved, mainly, one might surmise, because of the great difficulty of studying them in the living heart with the methods of modern hydrodynamics. In fact only since the development of the present interest in aerodynamics, ventilation, and the control of sound in modern house construction has been forced on engineers, has any new attempt been made to understand the factors of audible vibrations in moving fluids. The literature of the physics of hydraulic acoustics is strangely meagre in comparison with that of visible flow measurements. The doctor, however, cannot see the flow of the blood in the structures that he studies, but the noises that it makes are of dominant importance in the study of the heart.

No consideration of these auscultatory phenomena, however, can be intelligible without at least a caution as to the highly subjective character of such study. Audibility is a relative term. With special apparatus, for example, even without electrical amplification, the heart has been shown normally to have five sounds (Parker, 1918). The physician must ordinarily be content to hear three at best. Moreover, the human ear is very much more sensitive to high than to low pitched vibrations and no electrical device for recording sound vibration, unless corrected for this peculiarity, can expect to visualize what we hear. Again, most of us have areas in our auditory spectra that compare with blind spots in the eye, and are often dependent upon slight areas of degeneration in the mechanism of the ear from chronic upper respiratory infections. The training and experience of the observer play important parts in auscultation and some of the most useful auscultatory signs lie at the lower limit of human audition, where low pitch is about to change, at about 30 cycles per second, into a tactile and not an audible sensation.

The physicist has produced data and formulae of great significance for the theoretical analysis of heart murmurs, but only with difficulty can much of this be transferred to cardiovascular study. Such figures are based on the behavior of "ideal" or frictionless fluids, moving in straight tubes, in rigid systems, at constant velocities. The physician deals with a fluid of varying viscosity, in very complicated channels whose anatomy or pathology as regards the possibilities of stream-line, laminar, or turbulent flow is unknown. These channels, moreover, are elastic, and their contents move with an intermittent action.

But this transfer from hydrodynamics to cardiac physiology can be made so far as the known factors in the production of murmurs are concerned. The fundamental cause of sound in flowing fluids is the formation of turbulence by the intermittent building and release of eddies in the circulating medium. So long as the flow is stream-line, that is moving as a mass, and never at an angle to the tube, turbulence will not occur. In actual experience, as a result of friction at the surface of the tube and the production of regions of instability, turbulence will occur at very high velocities even in such a system. It is hard to imagine in the heart and vessels, with the repeated changes in caliber of the elastic vessels and chambers and the variations in cross-section from one part of the system to another, that the conditions of constant non-turbulent flow exist, except perhaps in peripheral vessels. But irrespective of whether or not turbulent flow is normally present in the heart, its occurrence does not necessarily imply an audible vibration transmitted to the external chest wall. Indeed calculations by Bondi, in Vienna, who has contributed much to this subject, would indicate that very high degrees of turbulence in blood vessels can exist before they can be heard. Such a finding would explain the presence or absence of some functional murmurs on the basis of variations in local sound transmission in individual patients.

The classical explanation of the production of audible eddies in the heart has stood since the days of Corrigan, Weber (1855), Chauveau (1858) and other early students of the subject. This, in brief, is that the most important mechanism is the passage

of a stream from an area of small caliber into one of larger cross-section. For example, one has only to constrict a rubber tube carrying water to observe the appearance of a murmur. Such a concept has the advantage of consistently and clearly explaining all murmurs produced within the circulation as dependent upon the same factors. Thayer (1925) in a very satisfying paper correlated murmurs on this basis and pointed out that the passage of the blood through the relatively smaller and more rigid aortic and pulmonic valve rings into the distensible vessels beyond was an excellent illustration of this mechanism and said that "it is not the frequency of soft systolic murmurs at aortic and pulmonary orifices that is remarkable. What is remarkable is that they are not always present," a statement he attributed to Broadbent.

While this escape of a jet of fluid into a vessel of larger caliber is no doubt the common mechanism for murmur production, the intricacies through which it passes as a sound vibration before its collection by the stethoscope have made many added factors worthy of consideration.

In general, *the conditions which are of primary importance in the birth of a heart murmur are these:*

(1) *The formation of jets, or discontinuous eddies, having vibration frequencies in the audible range.* A jet is formed whenever a narrow stream is projected at above a critical velocity into a gas or into a fluid at rest or with a different velocity from the entering stream. If the edges of the opening are sharp, as may be seen with rigid heart valves, eddies are set up with the greatest violence and the stream becomes extremely turbulent. If the expansion into the region of larger caliber is more gradual, or funnel-shaped, the turbulence is less and the vibrations are damped. At increasing velocities less change of caliber is needed to produce a murmur and a slight change will even cause a murmur at the entrance to the constricted area if the velocity is great enough.

(2) *The pressure gradient between the two sides of a constriction in the flowing stream which is passing into a chamber of larger caliber.* Such a pressure drop, of course, is the vital factor in the velocity. The presence of a velocity above a critical point is of the greatest importance in the appearance of a murmur. Bondi, in experiments on models, has found such a velocity to be in the neighborhood of 100 cm. per second with fluids of the same viscosity as blood. Velocity determines not only the appearance but the intensity of the murmur. It can readily be shown that murmurs can appear or increase when blood velocity is increased by exercise, and it can also be shown that, with reduction of pressure difference on the two sides of an obstruction, as in cardiac failure with aortic or mitral stenosis, loud murmurs will decrease or even disappear. Increased stroke volume such as occurs with exertion or excitement, anemia, aortic regurgitation and other conditions, will increase the velocity at the semilunar valve orifices and characteristically produce murmurs without the assumption of other than the physiologic stenosis of the ostia. The pressure change can, of course, be magnified by drop in peripheral resistance beyond the obstruction and thus velocity can be increased (Marey). In this way the murmurs of fever and hyperthyroidism can be explained on the same basis.

(3) *The object of impact of the jet.* A murmur becomes readily audible if the eddies formed by a jet can play directly upon the walls of the chambers of the heart, the blood vessels, or the damaged valves. This element of the impact of the jet upon a solid body is dependent upon the direction of the flow. In mitral regurgitation, for example, the stream strikes the posterior wall of the left auricle and the murmur produced may be soft and blowing at the apex but very loud and rough when observed with oesophageal auscultation. Sudden change of direction of the stream, as may occur normally in the pulmonary artery, plays a part in these impact phenomena and it is thought that jets against papillary muscles may also. Borgherini has described atrophy of an area in the anterior wall of the left ventricle in cases of aortic regurgitation at the point where the regurgitant stream impinges on the wall. This correlates with the known optimal position at the left sternal border for the discovery of aortic regurgitation.

While the fundamental conditions of the formation of a jet of sufficient velocity, causing eddies and impinging upon a sonorous body are commonly necessary for the production of audible vibrations, it can be shown experimentally that a murmur may be actually produced by a jet deep in the body of a fluid which, however, is inaudible unless the stethoscope be of the closed type and immersed to within one-half cm. of the upper border of the jet (Bondi). This again shows how other factors often must be present to make eddy currents enter the audible range both as to frequency and intensity. An added discussion of the determinants of "quality" or "timbre" would take us too far afield.

(To be continued)

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